

Chapter 6

Diseases of the Upper Respiratory Tract

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I. RHINITIS AND NASAL OBSTRUCTION

A. Rhinitis in horses

1. Patient profile and history
 - a. Acute rhinitis in the horse is most commonly found in young horses and is associated with infectious (viral, bacterial) respiratory diseases. In such cases, the infection is accompanied by signs of equine rhinotracheitis (see II A 3 f).
 - b. Fungal infections (cryptococcosis, rhinosporidiosis) also can cause rhinitis in the form of granulomatous, pedunculated masses. Cases are most common in the southern United States and are rare and sporadic. Tumors causing signs of rhinitis are uncommon in horses.
2. Clinical findings. Mucoid, mucopurulent, or blood-tinged nasal discharge is evident, as is inspiratory stridor if the nasal passages are markedly inflamed. In some cases, there may be decreased or unequal airflow from the nostrils, accompanied by malodorous air if necrotic processes are present.
3. Diagnostic and therapeutic plans (see II A 3)

B. Rhinitis and nasal obstruction in cattle

1. Etiology and pathogenesis
 - a. Causes include viruses, bacteria, fungi, allergens, and masses (tumors). Rhinosporidium-like organisms produce polyps in the anterior nares.
 - (1) Allergic rhinitis occurs mainly in Channel Island breeds, but it has been reported as a familial tendency in other breeds, occurring in spring and fall pollen seasons.
 - (2) Ethmoid carcinomas are sporadic causes of nasal obstruction. Although there may be a familial tendency for occurrence, there is also a viral origin implicated in the genesis of these tumors. The tumors are suggested to be of moderate malignancy and can metastasize to the lymph nodes and lungs.
 - b. Pathogenesis. Nasal obstruction may cause severe dyspnea, cyanosis, and stertorous breathing.
2. Clinical findings
 - a. Allergic rhinitis. Signs include acute dyspnea and sneezing, accompanied by yellow-orange nasal discharge. Chronic cases present with multiple nodules in the anterior nares. Affected animals may rub their nasal cavities by pushing sticks or twigs up the nostrils, resulting in lacerations or foreign bodies in the nasal cavity.
 - b. Ethmoid carcinomas. Signs include bulging facial bones, epistaxis, and dyspnea. The tumors are usually unilateral but can be bilateral, blocking both nasal passages. In these cases, open-mouthed breathing is often necessary. These tumors occur most often in older cattle, age 6–9 years.

C. Rhinitis and nasal obstruction in sheep and goats

1. Patient profile and etiology. Rhinitis and nasal obstruction in sheep and goats have primarily parasitic causes (e.g., *Oestrus ovis*) but can be sporadically caused by nasal tumors (e.g., adenopapillomas, adenocarcinomas) and viral infections or allergies, as in the other species. *Oestrus ovis* is the most common cause of nasal obstruction in sheep, but occasionally goats may also be affected.
2. Clinical findings include catarrhal to mucopurulent nasal discharge, sneezing, and difficult, snoring respiration.

3. Specific conditions

a. *Oestrus ovis*

(1) Etiology and pathogenesis

- (a) The fertilized females deposit larvae at the external nares during the summer and fall months. After hatching, young larvae crawl up the nasal cavity to the dorsal turbinates and frontal sinuses where they remain for several weeks to months before migrating to the nostrils and being sneezed out to pupate on the ground.
- (b) Irritation and secondary bacterial infection result in a purulent or mucoid nasal discharge, sneezing, low head carriage, and inspiratory dyspnea. Larvae can cause death in rare cases through secondary infections or encephalitis.
- (c) The presence of the adult fly attempting to larviposit on the nostrils can severely disrupt grazing; thus, the main effect of this parasite is production loss.

(2) Diagnostic plan. A definitive diagnosis can be made if the larvae are seen in the nasal cavity; however, a careful search for foreign bodies in the nasal cavity should be made.

(3) Therapeutic plan and prevention. Effective antibiotics include ivermectin (200 $\mu\text{g}/\text{kg}$) orally or raxofanide 7.5 mg/kg (where available). Treatment should be administered in late summer to prevent buildup of heavy infestations. Winter treatment removes overwintering larvae.

b. Miscellaneous causes

- (1) Allergic rhinitis
- (2) Bluetongue

- (3) Contagious ecthyma (see also Chapter 11 | 9) usually is confined to the mouth and external nares but may spread to the muzzle and nostrils and, in rare cases, to the upper and lower respiratory tract, resulting in bronchopneumonia.
- (4) Nasal adenopapillomas are not uncommon in mature animals.
 - (a) Enzootic nasal adenocarcinomas occur in sheep in the United States and Canada where the incidence in individual flocks suggests an infectious cause. A similar-appearing tumor is enzootic in goats in Europe, where extracellular retroviral-like particles have been observed. A similar tumor is only rarely reported in goats in North America.
 - (b) Clinical findings include nasal stridor and dyspnea, with progressive anorexia. The tumor is only locally invasive and not metastatic. However, because of the location, death from asphyxiation or inanition usually occurs within 90 days from the onset of clinical signs.

D. Rhinitis in swine

1. Atrophic rhinitis

- a. Patient profile and history. This disease affects mostly young pigs but the residual anatomical defects persist for the life of the pig. The condition starts as an episode of acute rhinitis, followed by chronic atrophy of the turbinate bones.
- b. Clinical findings. This disease is characterized by shortening or distention of the snout, sneezing, nasal discharge, and epistaxis in growing pigs. Severe cases may exhibit impaired growth rates.
- c. Etiology and pathogenesis. There is substantial evidence to implicate *Bordetella bronchiseptica* as the inciting agent of the acute inflammation, followed by invasion of toxigenic strains of *Pasteurella multocida*. Up to 50% of finished pigs may have evidence of atrophic rhinitis. The true economic significance of atrophic rhinitis remains undetermined, as field studies have failed to show strong evidence of adverse effect on daily weight gains in growing pigs.
- d. Prevention has been attempted with early administration of antibiotics (e.g., tylosin, oxytetracycline, trimethoprim-sulfadiazine) in the early creep feed. Control has been aimed either at total eradication by depopulation or reduction of infection through mass medication or vaccination of pregnant gilts with *B. bronchiseptica* followed by *P. multocida* bacterins.

- 2. Inclusion body rhinitis. This common disorder is caused by a β -herpes virus but is fortunately a minor disease in young pigs. Occurrence is probably worldwide, clinically affecting piglets up to 10 weeks in age. Sneezing is the most prominent sign, often occurring in paroxysms following play fighting. There may be a mild serous nasal discharge, occasionally blood tinged, with all the piglets in the group affected. The clinical course is usually 2–4 weeks with no mortality. This virus is not implicated in the genesis of atrophic rhinitis.
- 3. Swine influenza
 - a. Patient profile and history. This highly contagious disease is characterized by fever and watery ocular and nasal discharge in a high proportion of the herd.
 - b. Clinical findings and etiology. The disease is caused by the type A influenza virus, possibly from an adaptation of the human influenza virus. Pigs also develop anorexia, prostration, and labored jerky breathing (thumps), accompanied by sneezing and a deep painful cough, often in paroxysms. Often, after 4–6 days, the signs rapidly abate.
 - c. Therapeutic plan and prevention. There is no specific treatment, and although vaccines have been produced, the antigenic diversity of the virus may limit effective immunity.
- 4. Necrotic rhinitis. Otherwise known as bullnose, this disease is characterized by facial deformities and is often confused with atrophic rhinitis.
 - a. Patient profile and history. This condition is most commonly seen in growing pigs that are raised in poor environments with heavy organic debris contamination.
 - b. Clinical findings. Initially, there is cellulitis of the soft tissues of the nose and face with localized swelling that may interfere with respiration and mastication.
 - c. Etiology and pathogenesis. *Fusobacterium necrophorum* is commonly isolated from the affected sites. If untreated, the inflammation spreads to the nasal bones and can cause facial deformity, toxemia, reduced appetite, and death.
 - d. Differential diagnosis. The main differentiating feature of bullnose in comparison to atrophic rhinitis is the presence of soft tissue cellulitis, which is usually completely lacking in atrophic rhinitis.
 - e. Therapeutic plan and prevention. Antibacterials, such as sulfonamides, are effective when treating young infected pigs and early stages of the disease. However, the aim should be the reduction of incidence, which is best managed by improved sanitation and disinfection of pens and elimination of any material that may cause mouth or head injuries (sharp edges on feeding troughs or waterers).
- 5. Pseudorabies. Some outbreaks of pseudorabies (Aujeszky's disease) may show signs of rhinitis.

II. ACUTE PHARYNGITIS, LARYNGITIS, AND TRACHEITIS

A. Pharyngitis, laryngitis, and tracheitis in horses

- 1. Etiology
 - a. Infectious agents are the most common clinical causes of important diseases with predominately upper respiratory signs.
 - b. Noninfectious causes are more sporadic and include irritation, pharyngeal abscess, foreign body, or retropharyngeal lymph node rupture.
- 2. Clinical significance
 - a. Decreased exercise tolerance. It is often necessary to halt racing and showing activity in horses with upper respiratory tract infection.
 - b. Secondary infection. These disorders predispose the horse to secondary bacterial infections of the lower respiratory tract. Only rarely do these viruses cause lower respiratory disease, with the exception of immunocompromised animals (see II A).

3 e). Although viral respiratory disease commonly causes only transient mild pyrexia in most animals, it can be fatal in young animals.

3. Specific conditions

a. Equine influenza virus infection is one of the most common causes of viral respiratory disease in horses older than 2 years.

(1) Patient profile and history

(a) Affected horses are primarily the young stock (age 2–3 years), but all ages are susceptible. Older horses usually have a milder infection or may show few signs other than a transient fever.

(b) The disorder usually occurs as an explosive outbreak of respiratory disease in stables, but where immunity is strong, either from past exposure or vaccination, signs may be limited to several horses in a stable showing only fever or mild hindlimb edema. Less commonly there can be associated complications of myositis and myocarditis.

(2) Epidemiology

(a) A hallmark of this infection is its extremely rapid spread through a population of susceptible horses. In contrast to the shorter surviving human and swine types of virus, the equine type virus survives for up to 36 hours on fomites. This, combined with a short incubation period and a 3- to 8-day period of infectivity of affected horses, produces a rapid new infection rate.

(b) Outbreaks often occur in spring and fall. Risk **factors** include:

- (i) A mixing of young horses, such as in the show or racing season
- (ii) An increased number of unvaccinated animals
- (iii) Stress caused by moving and crowding

(c) The reservoir for this virus is unknown, but there may be inapparent carriers or vaccinated, asymptomatic carriers. Equine influenza is common in most countries, with the exception of Australia and New Zealand, where it has yet to be recorded.

(3) Etiology and pathogenesis

(a) The causative virus is a myxovirus, which is an RNA virus with two serologically distinct antigenic types (influenza A/equi 1 and A/equi 2). There seems to be no cross-species infection, and, as in most influenza viruses, the viruses are subject to antigenic drift. Thus, the commercially available vaccines, although affording protection, are seldom 100% effective.

(b) Infection is initiated by inhalation or **contact** with nasal secretions from an infected animal. The virus can persist in an infected horse's secretions for up to 8 days, and the most common source of infection is coughed secretions.

(c) After an incubation period of 1–5 days, the **infection** results in clinical signs, which reflect the epithelial inflammation of the respiratory tract. When the virus invades the respiratory epithelium, changes include **hyperemia**, edema, and cellular desquamation. Superficial erosions to the upper and lower respiratory tracts can occur with a loss of normal **mucociliary** clearance mechanisms, which provide at least transient potential for secondary bacterial invasion.

(d) **Postinfection** complications

(i) When the virus has cleared, the respiratory epithelium can take up to 3 weeks to fully recover to its normal state. This may be one reason that some horses continue to exhibit coughing for **several weeks** after apparent resolution of the infection.

(ii) Associated but less common complications include myocarditis with arrhythmias (atrial fibrillation), secondary bacterial pneumonia, pleuritis, persistent cough, and exacerbation of underlying chronic obstructive pulmonary disease.

(4) Clinical findings

(a) The disease causes a fever (38.5°C–41°C) and a dry hacking cough,

which later turns moist and persists longer than the fever. Nasal discharge is watery when present but is seldom prominent.

(b) Adenopathy. Although the submaxillary lymph nodes are not appreciably swollen, they are often painful to palpation in the early stages, which indicates the pharyngeal inflammation that occurs and may cause signs of dysphagia.

(c) Other signs include dyspnea with or without exercise, systemic signs associated with infection (e.g., fever, inappetence, lassitude), or muscular stiffness and limb edema. In uncomplicated cases, the signs usually resolve completely within 3 weeks.

(5) Diagnostic plan and laboratory tests

(a) On the routine complete blood cell count (**CBC**), there can be a leukopenia with distinct lymphopenia, but this is transient. For a definitive diagnosis, virus isolation is necessary.

(b) Nasopharyngeal swabs must be collected in the first 48–72 hours of illness, beyond which viral culture is unlikely to be successful.

(c) Serologic confirmation of infection relies on a rise in antibody titer in paired sera collected 3 weeks apart, with a positive finding based on a fourfold rise in hemagglutination inhibitor or serum neutralization titer.

(d) For rapid diagnosis, a test based on direct immunofluorescence applied to nasopharyngeal smears also has been successful in investigating outbreaks.

(6) Differential diagnoses

(a) **Viral infections.** This includes mainly the other upper respiratory viral infections (herpesvirus, rhinovirus, or adenovirus). Although equine viral arteritis (EVA) also can cause respiratory signs, these signs are often of secondary importance. Other signs, such as conjunctivitis, petechiation of mucous membranes, and limb and palpebral edema, are more prominent.

(b) Bacterial infection by **Streptococcus equi** also can cause similar initial clinical signs, but generally the submandibular lymph nodes are obviously enlarged and painful.

(7) Therapeutic plan

(a) As with most viral infections, there are few specific treatments available to hasten recovery. The main goal is to provide a clean, stress-free environment to allow the horse to recover from the infection.

(b) For increasing the comfort of the horse, nonsteroidal anti-inflammatory drugs (**NSAIDs**), such as phenylbutazone, can be used to decrease fever and maintain the horse's appetite during the acute phase of the infection. However, disadvantages of this approach include:

(i) The antipyretic action of **NSAIDs** might mask any fever due to secondary bacterial infection.

(ii) Owners might return the horse to competition or work before the effects of the disease have fully abated.

(c) Antibiotic treatment is appropriate if secondary bacterial infection is suspected or in high-risk animals, such as young foals. Ideally, the choice of drug should be based on culture results from transtracheal wash. In the absence of this, broad-spectrum antibacterials such as **trimethoprim-sulfas** can be administered, with a course when initiated of 5–7 days.

(8) Prevention

(a) **Vaccines.** There are several manufacturers of killed-strain influenza vaccines, containing both A/equi 1 and A/equi 2. Manufacturers recommend two intramuscular injections several weeks apart initially, then revaccination annually.

(b) Reaction to vaccination. Vaccinated horses should be rested for several days after vaccination because they are often reported to be "off" the day following injection. Horses develop a transient reaction to vaccination that may include mild fever, malaise, and pain at the injection site.

(c) Frequency of vaccination. Vaccination results in at least partial immunity

to disease, but not to infection. The duration of immunity from any of the vaccines is probably less than 1 year. Vaccination in the face of an outbreak may be beneficial if it can be done in advance of the spread of disease.

- (i) For high-risk animals, such as young horses in the show season, it is recommended to repeat vaccination every 3–4 months during the high-risk period.
- (ii) For backyard horses with no new additions, vaccination may not even be necessary for those horses older than 3 years.
- (iii) For foals, the usual recommendation is to begin vaccination between 2 months and 6 months. Some workers suggest that beginning vaccination at 30 days of age may decrease the incidence of foal pneumonia.

b. Equine viral rhinopneumonitis [equine herpesvirus (EHV) 4 and 1] infection

- (1) **Patient profile and history.** Rhinopneumonitis usually is most prominent in weanlings and yearlings that are experiencing periods of intermingling and stress. However, this disease can be seen in horses of all ages, with the presenting complaints being fever, conjunctivitis, and coughing.
- (2) **Epidemiology**
 - (a) Carrier animals are often present in herds and serve to maintain the infection from year to year. The disease can be reactivated in periods of stress or by corticosteroid administration.
 - (b) Rapid spread through a herd is associated with high morbidity and low mortality rates; the respiratory disease is generally mild. Outbreaks most commonly occur in fall and winter months, and 85% of respiratory outbreaks involving EHV in serologic surveys are attributed to EHV-4.
- (3) **Etiology and pathogenesis**
 - (a) **Etiology**
 - (i) The disease is caused by several differing strains of EHV, with EHV-4 causing most of the outbreaks of respiratory disease in horses in any population at any time of the year.
 - (ii) EHV-1, serologically related to EHV-4 by only 20% homology, has two subtypes. Subtype 1 is associated with abortion. Subtype 2, while also abortigenic, can cause respiratory disease.
 - (iii) **Neurologic disease** can also accompany EHV-1 infection, but the pathogenesis is poorly understood (see Chapter 11).
 - (b) **Pathogenesis**
 - (i) This highly infectious disease is transmitted by inhalation or contact with secretions (e.g., placenta, nasal secretions, aborted fetuses) containing infectious EHV particles. However, because the virus can survive from 15 to 45 days outside the animal in the environment, infections can occur in the apparent absence of an initiating case.
 - (ii) The virus rapidly proliferates in the mucosa of the nasal, tonsillar, and pharyngeal regions, resulting in the rhinitis, pyrexia, and associated respiratory signs. Following this, there is a short-lived viraemia in which the virus is closely associated with circulating lymphocytes, from which the virus can be isolated. The virus is then transported to the tissues (e.g., lung, placenta, fetus, nervous tissues) and induces various subsequent organ damage.
 - (iii) **Immunity.** The virus can be present in nasopharyngeal swabs for up to 10 days and can be shed spontaneously at times of stress in carrier animals. Immunity to these forms of herpesvirus is weak, and an animal can become clinically affected several times. Passive immunity in foals in the form of antibodies declines to zero by 180 days after birth. However, even the presence of virus-neutralizing antibodies are not necessarily an indication of resistance to infection, as cell-mediated immunity is a key feature of resistance to herpesvirus.

(4) Clinical findings

- (a) The respiratory signs vary with the amount of exposure, animal age, and

immunity. Signs are similar to those of influenza but are milder and more transient. As with influenza, younger animals most obviously are affected in an outbreak, whereas older animals may show few or no signs of respiratory disease.

- (b) The most common findings are pyrexia (39.5°C–40.5°C), conjunctivitis, serous nasal discharge, and a possible mild cough. The appetite of infected horses generally remains unaffected, and there may be slight enlargement of lymph nodes or the throat region.
- (c) The clinical course is usually 3–7 days, but some horses may cough for up to 3 weeks. Whereas some horses may undergo inapparent infection, very young foals can develop a primary interstitial pneumonia. Reinfection may occur within 4–5 months, but usually immunity from clinical infection lasts 6–12 months.
- (d) Endoscopic examination of the upper respiratory tract shows mild mucosal inflammations, consisting of rhinitis, pharyngitis, and lymphoid hyperplasia.
- (e) For respiratory disease caused by EHV-1, there may also be **abortions** up to 4 months later and possibly **neurologic disease** 8–11 days after respiratory infection on the same farm.
- (5) **Diagnostic plan.** Respiratory disease accompanied by abortions, neurologic signs, or both is strong presumptive evidence of EHV-1 infection. However, where respiratory signs are the only abnormality, confirmation of infection can be either by acute and convalescent serology, or by virus isolation from nasal secretions, which is possible for up to 10 days.
- (6) **Laboratory tests.** On the routine E6E, there is, as in most viral diseases, a nonspecific leukopenia and lymphopenia. Confirmation of the infection is best accomplished by collection of acute and convalescent titers, but virus isolation can be successful from nasal washing obtained in first few days of infection.
- (7) **Differential diagnoses.** For problems that are solely showing signs of upper respiratory disease, the same considerations as for influenza are appropriate. These include equine rhinovirus, adenovirus, and viral arteritis. Bacterial infection by *Streptococcus equi* also can show similar initial clinical signs, but generally the mandibular lymph nodes are obviously enlarged and painful.
- (8) **Therapeutic plan.** As for influenza and other upper respiratory infections, there is little specific treatment other than providing a clean, draft-free, low-stress environment and discontinuing work while the horse is allowed to recover.
- (9) **Prevention is with vaccination.** Because the respiratory disease is sufficiently mild, a major goal of vaccination is the prevention of abortion. It is generally accepted that vaccination should be incorporated into routine health maintenance only when there is a known enzootic problem.
 - (a) There are cell-culture-adapted **live viruses** that can be used safely for routine work in most breeding farms; however, the weakness is the brevity of immunity from this vaccine. Brood mares need to be vaccinated twice during the latter half of pregnancy, and its use in foals and yearlings requires, at the minimum, trimonthly boosters.
 - (b) A killed vaccine is in wide use for protection against abortion due to EHV but should not be assumed to protect against respiratory signs.
 - (c) To protect against the **respiratory form** of the disease, a modified live EHV-4 combined with a EHV-1 and influenza antigens is available. This vaccine is administered initially when a foal is 2–4 months of age, repeated in 4–8 weeks and then yearly until 2 years of age. Many clinicians recommend more frequent administration of these antigens in areas where the incidence of clinical disease is high.
- (c) **Equine viral arteritis (EVA)**
 - (1) **Patient profile and history.** EVA, a disease seen in outbreaks on breeding farms, causes pregnant mares to abort. Serological evidence suggests a high

prevalence of exposure (70%–90%) in Standardbreds, but Thoroughbreds have only a 2%–3% seropositive rate.

- (2) **Etiology.** EVA is caused by an arterivirus similar to the agent that is implicated in porcine reproductive and respiratory syndrome. Virulence between strains varies, but there is little antigenic variation.
- (3) **Clinical findings**
 - (a) This disease primarily causes abortion, with acute systemic illness including fever, limb edema, and respiratory signs. Abortion usually occurs within a few days of clinical onset of disease. This is in contrast to abortions caused by equine viral rhinopneumonitis, which occur much later after clinical disease.
 - (b) The respiratory signs are usually of secondary importance to the disease and include nasal and ocular discharge that is initially serous but can become purulent. There is a cough, nasal mucosal congestion, and in some horses, petechiation. In horses with pulmonary edema, dyspnea also may occur.
- (4) **Pathogenesis.** Although outbreaks of the classic disease can occur, infections are commonly subclinical with sporadic abortions. Stallions can act as carriers, and there is an effective vaccine available for prevention where indicated.
- (5) **Therapeutic plan and prevention**
 - (a) Specific therapy for this viral infection is not available, so treatment is only directed against secondary bacterial infection if respiratory signs are present.
 - (b) Quarantine. Because the virus is contagious, the quarantine of any infected horse returning from a racetrack, sale, or show may be necessary for up to 4 weeks.
 - (c) A currently available modified-live virus vaccine may be useful to control this disease on breeding farms.
- d. **Equine rhinovirus (ERV) infection**
 - (1) **Etiology.** Infection with rhinovirus, equine rhinovirus-1 (ERV-1), is widespread, and most of the population of horses develop antibodies early in life. Infection with several types of rhinovirus (ERV-1, ERV-2, and ERV-3) is common, but only ERV-1 is clinically significant.
 - (2) **Clinical findings.** Although infection is often subclinical, horses with clinical signs exhibit low-grade fever, pharyngitis, pharyngeal lymphadenitis, and copious serous nasal discharge that becomes purulent in later stages. A cough may persist for 2–3 weeks.
 - (3) **Diagnostic plan.** Serum neutralization titers from acute and convalescent sera can establish a diagnosis. Alternatively, virus isolation may be attempted to allay concerns regarding more significant problems associated with influenza or rhinopneumonitis.
 - (4) **Therapeutic plan and prevention.** Because clinical disease is usually mild and self-limiting, there is little that needs to be done for treatment other than appropriate rest to allow recovery. Currently, there is no commercially available vaccine; however, planned exposure of young horses to infection has been recommended.
- e. **Adenovirus infection**
 - (1) **Patient profile and history.** Adenovirus infection in the horse population appears to be widespread but is clinically of major importance in the Arabian breed.
 - (2) **Clinical findings.** Infection with adenovirus in adult horses generally does not cause any clinical disease. However, mild respiratory signs and transient softness of the feces may result in some cases. The exception to this usually mild infection is infection of immunocompromised foals, such as in failure of passive transfer or Arabian foals with inherited combined immunodeficiency (CID). Experimentally, this virus can cause a severe but nonfatal pneumonia, with the full scope of signs including conjunctivitis, coughing, nasal discharge, fever, dyspnea, and diarrhea. In Arab foals with CID, the pneumonia

is invariably fatal. There is hyperpnea, dyspnea, and adventitious sounds on auscultation of the lungs, suggestive of bronchopneumonia. Affected foals often have a poor hair coat, are depressed, and may have diarrhea.

- (3) **Etiology and pathogenesis.** Adenovirus infection is widespread in all ages of horses. Arabian CID foals develop fatal pneumonia and die, regardless of therapy. The virus attacks the upper and lower respiratory epithelium and leads to loss of epithelium with subsequent hyperplasia and interstitial pneumonia. Secondary bacterial bronchopneumonia also ensues, but these foals lack the ability to mount any defenses to recover from infection.
- (4) **Diagnostic plan.** For most horses, the infection is mild, and diagnosis can be made from a number of serologic tests or from identification of the characteristic intranuclear inclusion bodies in cells taken from conjunctiva or nasal mucosa.
- (5) **Laboratory tests and differential diagnoses**
 - (a) In the case of an affected Arabian foal, it is important to exclude CID. Tests include the presence of lymphocytes above 1000 μ l, and the presence of precolostral serum immunoglobulin M after 36 days of age.
 - (b) Other viruses, including equine parainfluenza-3 (PI-3) virus and a reovirus, have also been recorded as viruses to be suspected in causing mild upper respiratory tract inflammation in horses but are of doubtful significance.
- (6) **Therapeutic plan and prevention**
 - (a) For adult **horses**, there is no specific treatment other than as indicated for other mild upper respiratory infections.
 - (b) Foals with failure of passive transfer are at increased risk of adenovirus infection because it is so prevalent in the horse population. These foals should receive plasma and, in high-risk situations, antibiotics.
 - (c) Although an inactivated vaccine of apparent high immunogenicity has been produced against adenovirus in the horse, it has not had extensive field exposure.
- f. **Noninfectious** equine rhinotracheitis can be the result of pharyngeal abscess, foreign body, irritation from orally administered medication, or **nasogastric intubation**. These causes are far less common than infectious causes.
- g. **Chronic pharyngitis.** The most commonly managed chronic problem of the pharynx is lymphoid hyperplasia (**follicular pharyngitis**), as observed by endoscopy in performance horses. This abnormality does not appear to affect health but is suggested by some to affect athletic ability. This and other pharyngeal abnormalities, including structural or functional abnormalities of the soft palate or larynx, are in the realm of equine surgery.

B. Acute pharyngitis, laryngitis, and **tracheitis** in cattle

1. **Clinical findings.** The common upper respiratory tract infections in cattle frequently have nonspecific signs, and an etiologic diagnosis based solely on clinical signs is not possible. The exception to this is infectious bovine **rhinotracheitis** (IBR), in which there can be characteristic nasal reddening, plaques, and conjunctivitis.
2. **Pathogenesis.** Most of the incriminated bovine respiratory viruses are ubiquitous, and exposure and immunity are widespread. An important factor of these infections is the potential to compromise **normal** pulmonary defense mechanisms and allow colonization by bacteria that would normally be cleared. Therefore, although viral **respiratory** infections may not extend to the lower respiratory tract, secondary bacterial infection is the primary consideration in the management of both treatment and prevention.
3. **Diagnostic plan and laboratory tests.** Specific etiologic diagnosis requires virus **demonstration** or isolation and identification and is seldom successful in later stages of the disease. To maximize the potential of an etiologic diagnosis, a routine set of **samples** should be obtained in outbreaks.
 - a. From live, acutely infected febrile untreated **cattle**, nasal **swabs** or conjunctival scrapings are suitable for most viral isolation. Acute and convalescent serum **samples** also can be collected and screened for the suspected viruses, with a fourfold

titer increase over 2–3 weeks proving active recent infection. For some viruses (e.g., bovine respiratory syncytial virus), immunofluorescence on lung lavage cells also has proven highly valuable.

b. Necropsy of animals can be useful but is seldom rewarding in chronically ill, treated, or cull animals or those dead long enough to have undergone significant organ autolysis. In a field necropsy, tissues selected for virus isolation include conjunctiva, tonsil, pharynx, lung, lymph node, spleen, liver, kidney, small intestine, cecum, spiral colon, and rectum. These tissues require placement in appropriate viral transport medium, freezing, and transport by overnight courier to the laboratory.

4. Therapeutic plan

- Supportive care including ready access to feed and water and minimizing competition for feed and space is often all that is required.
- Antibiotic therapy for possible secondary bacterial infection can be instituted. Although glucocorticoids impair defense mechanisms and are contraindicated in IBR infections, NSAIDs (e.g., aspirin) may be beneficial in reducing fever and improving appetite. Therapy of individual animals is generally not as important as control of herd outbreaks.

5. Prevention. Vaccines are available for the more clinically important viruses and are often combined in a single product.

- Modified live virus of bovine cell origin for IBR vaccines should not be used in pregnant cows because these vaccines may cause vaccine virus shedding, infertility, and abortions. Potential stud bulls should be vaccinated with products containing the IBR antigen only at the owner's request.
- Products that are designed for intranasal administration of live vaccine stimulate strong local (immunoglobulin A) immunity and a rapid response, including local interferon.
- When considering the young stock in the herd, any early vaccination, such as is sometimes performed by administration of intranasal vaccines soon after birth, should be repeated after 2 months of age because the colostral immunity has waned by that time for most of these viruses.
- In dairy herds, annual or semiannual vaccination is recommended, as well as vaccinating any replacement animals 3–4 weeks before introduction.
- In feedlot management, because of high stress of transport and shipping, killed or at least inactivated vaccines may be appropriate, unless modified live viruses can be administered at least several weeks in advance of anticipated stress periods.

6. Specific conditions

- Infectious bovine **rhinotracheitis** (IBR), also known as bovine herpes virus-1 (BHV-1), is a highly infectious condition of worldwide distribution in cattle and some wild ruminants. Recent studies have revealed at least five major biotypes, which might explain the distinct clinical manifestations of this viral infection. The respiratory form is a common manifestation of infection in cattle and is usually restricted to upper respiratory signs, as uncomplicated IBR does not usually spread into the lungs.
 - Patient profile and history. BHV-1 can affect all ages of cattle, but those older than 6 months are most commonly affected, with animals in beef feedlots experiencing higher morbidity than dairy herds.
 - Epidemiology. The morbidity rate varies from 8% to 20%–30% in feedlots, but in herds of low immunity, morbidity can approach 100%. However, the case fatality rate is usually low at 1% or less. Where fatality rises to 10%, deaths are usually related to secondary bacterial bronchopneumonia (see Chapter 7).
 - Etiology and pathogenesis
 - Transmission. The α-herpes virus BHV-1 is transmitted via respiratory aerosol, semen, fetal fluids and tissues, and fomites.
 - After infection in the field, it appears there is an incubation period of 10–20 days, although experimentally this only lasts 3–7 days. The BHV-1 virus multiplies in nasal mucosa, causing rhinitis and tracheitis. Viral ex-

tension via the lacrimal duct causes conjunctivitis and, in some cases, corneal edema.

(c) Route of infection. From nasal mucosal infection, the virus travels up the trigeminal nerve, where it can establish latency or infect the central nervous system. The virus also can be transported by peripheral leukocytes to the placenta and transferred to the fetus, resulting in abortion.

(d) Carriers

- The virus can persist and be discharged from the animal as a result of natural infection or live virus vaccination for at least 2 years.
- Carrier states are thought to occur in some cattle, and there also may be wildlife reservoirs of infection in wild ruminants.
- Latent infections occur with the virus presumably sequestered in the trigeminal ganglion, which can recrudesce during stress or corticosteroid administration.

(e) Immunity to BHV-1 is complex and requires both cell-mediated and humoral parts of the animal's defenses. Therefore, it follows that systemic antibody levels, as determined by the many serologic tests available, correlate poorly with protection against disease.

(4) Clinical signs

- The various clinical manifestations of IBR include:
 - Upper respiratory inflammation with or without prominent conjunctivitis
 - Venereal infections, such as infectious pustular vulvovaginitis (IPV) in cows and balanoposthitis in bulls
 - Encephalitis
 - Infertility
 - Abortion
- With the respiratory form of IBR, there is a sudden onset of severe signs, including high fever (up to 42°C), anorexia, and severe hyperemia of the nasal mucosa.
 - Necrotic plaques appear as greyish foci of necrosis on the nasal mucosa just inside the nares, accompanied by a serous to mucopurulent nasal discharge.
 - Conjunctivitis with a serous ocular discharge is also a common sign. Although this may sometimes be mistaken for infectious keratoconjunctivitis caused by *Moraxella bovis*, the lesions are confined to conjunctiva, which are reddened and swollen with no invasion and ulceration of the cornea.
 - A short, explosive cough can accompany these signs but is not always present in outbreaks. If lactating dairy cattle are affected, there is also an accompanying dramatic fall in milk production.
- Duration of infection. When restricted to respiratory signs, infection generally resolves in 10–14 days. Abortions can occur some weeks following clinical illness. The genital tract infections (IPV or balanoposthitis) can result in reproductive failure.
- Diagnostic plan and differential diagnoses. The classic signs of fever, nasal lesions, and bilateral conjunctivitis should suggest the respiratory form of IBR. Other possibilities include:
 - Bovine respiratory disease complex (BRDC; shipping fever). With BRDC, there is toxemia, abnormal lung sounds, and affected animals respond well to antibiotics.
 - Bovine virus **diarrhea/mucosal** disease (BVD/MD). With BVD/MD, there should be oral erosions and usually diarrhea in addition to nasal ulceration.
 - Bovine malignant catarrh (BMC). BMC exhibits similar signs to BVD/MD.
 - Calf diphtheria. This condition may resemble IBR with inspiratory dyspnea but usually has severe toxemia and necrotic oral and laryngeal lesions.
 - Allergic rhinitis. Although it resembles IBR, allergic rhinitis does not

result in fever and usually is accompanied by sneezing and a characteristic thick greenish orange nasal discharge.

(6) Therapeutic plan

- Antibiotics. Although of no direct effect against the viral infection, antibiotics such as oxytetracycline or sulfa drugs can be given to control against secondary bacterial tracheitis and bronchopneumonia. As most cattle recover uneventfully without antibiotics, this must be weighed against the cost of treatment and possible need for appropriate withdrawal periods of milk or meat.
- Management strategies. As in other viral infections, it is important to aid recovery by reducing stress (e.g., crowding) and providing high-quality feed and access to water. In feedlot situations, this is best managed in a separate "sick pen" where competition for feed and space is reduced, and particular attention can be given to monitor for signs of onset of secondary bronchopneumonia.
- Glucocorticoids** are specifically contraindicated in this disease.

(7) Prevention

- There are several effective vaccines commercially available. Because the disease can occur unpredictably at any time and even in what seem to be closed herds, vaccination by either an intranasal aerosol or modified live vaccine or intramuscular modified live or inactivated vaccines is indicated.
 - The intranasal vaccines stimulate local as well as humoral immunity, and in addition to being safe for use in pregnant cows, can be used in the face of an outbreak because of stimulation of local interferon within 72 hours of administration. However, these vaccines are more labor intensive to administer and generally more expensive.
 - Intramuscular vaccines, if not inactivated, can cause abortion and infertility. Thus, if used, they should be given to heifers and cows at least 2 weeks before breeding.
 - Vaccination with modified live **products**, although stimulating a stronger immune response than that obtained from inactivated products, may result in shedding of the virus.
- For those herds in which export of cattle or production of bulls for artificial insemination units is an economically important consideration, vaccination against BHV-1 is not advised because this may result in the rejection of animals for export or sale to bull studs.

b. Bovine adenovirus. This virus often causes an inapparent infection, and although a potential cause of upper respiratory inflammation, it is relatively unimportant other than for its possible association with pneumonia, enteritis, or both in calves. Adenovirus may play a role in enzootic calf pneumonia (see Chapter 7). Adult cattle are the source for infection in calves. Latent infection with stress may result in recrudescence and viral shedding.

c. Bovine viral diarrhea (**pestivirus**, BVD). Similar to adenovirus, BVD can result in mild, nonspecific clinical signs of respiratory infection. Although BVD virus is not a primary pathogen of the respiratory system, some affected animals do have oral lesions extending into the nasal cavity and signs suggestive of primary rhinitis. However, oral erosions and gastrointestinal signs such as diarrhea are usually striking and avoid confusion with uncomplicated rhinitis and upper respiratory tract infections. A main factor in the relationship of BVD virus to respiratory disease is the immunosuppressive effects of infection (see Chapter 7).

d. Parainfluenza-3 (PI-3)

- Patient profile and history. PI-3, a paramyxovirus, is a ubiquitous virus that can be recovered from normal and acutely ill cattle. The major importance of this infection is its potential link to pneumonia in calves (see Chapter 7) and bacterial bronchopneumonia in older cattle.
- Clinical findings. Animals with uncomplicated infections have mild upper re-

spiratory disease with cough, serous nasal discharge, and fever. The clinical abnormalities are nonspecific for this infection.

(3) Pathogenesis

- The virus invades the respiratory tract and can infect pulmonary **macrophages**, which may permit viral replication and impair phagocytic function.
- PI-3 infection has been associated with concurrent viral infections (e.g., bovine respiratory syncytial virus) or *Pasteurella haemolytica* infection and serious pneumonia.

(4) Diagnostic plan. Acute and convalescent serology can establish the occurrence of recent infection, and the virus can be isolated from affected animals.

(5) Therapeutic plan and prevention

- As in the other mild upper respiratory infections, this is usually **self-limiting** and needs no treatment unless signs of pneumonia occur with secondary bacterial infection.
- There are a number of commercially available vaccines that contain this antigen. For calves, a modified live intranasal vaccine stimulates both nasal and humoral antibodies and appears effective against challenge exposure.
- The antigen is also commonly included in **multivalent** vaccines for prevention of undifferentiated bovine respiratory disease.

e. Bovine **respiratory syncytial virus** (BRSV)

(1) **Patient profile and history**

- Infection by BRSV can occur in any age of cattle, particularly in immunologically naive herds. Beef breeds may be more susceptible than dairy animals. There is generally rapid onset resulting from a short incubation period and rapid spread of the disease through the herd. Fall and winter are the most common times for disease.
- Although it causes upper respiratory signs (e.g., serous nasal discharge, cough), this viral infection is more **important** in its ability to invade the lung and cause viral pneumonia (see Chapter 7). A high percentage of North American cattle have serum antibodies to BRSV, indicative of widespread (greater than 80%) infection. Conversely, clinical disease is not nearly so common. The virus has a close antigenic relationship to sheep and goat RSV as well as human RSV.

(2) **Clinical findings** vary depending on the stage of the disease, primary or secondary.

- Primary stage. There is sudden onset of fever (40°C–42°C), **nasal/lacrimal** discharge, cough, hypersalivation, and decreased appetite. Tachypnea (more than 30 breaths/min) and abnormal lung sounds (i.e., increased bronchial tones, wheezes, fine crackles) also may be present, indicating viral invasion of the lungs. If infection occurs in lactating cows, there is a moderate but transient fall in milk production. A transient diarrhea also may occur. In this stage, clinical signs are usually mild and quickly resolve.
- Secondary stage. The term **secondary stage** arises from the clinical course of such animals often appearing after remission from an initial mild infection of BRSV and the suggestion that this manifestation may be a hypersensitivity reaction to the initial antigen, with involvement of immunoglobulin E (see Chapter 7).
 - In the secondary stage, the disease produced is far more severe, with **profound dyspnea**, anorexia, and open-mouth breathing with froth at the muzzle. These animals are often unable to eat because of severe dyspnea. Some animals have subcutaneous emphysema **over** the bunk and submandibular edema.
 - Auscultation of the chest often reveals prominent crackles. These animals are clearly in respiratory failure.

(3) Pathogenesis. Despite considerable research efforts, the pathogenesis of BRSV infection remains poorly understood. Experimental infection has been difficult

to achieve and, in most cases, results in only mild clinical disease with limited lesions.

- The virus infects respiratory tissues from the nasal cavity to the bronchi/bronchiolés. Epithelial destruction follows and peaks at 5–7 days post infection but is usually mild and incomplete.
- The rapid serologic response (3 days) makes routine serologic testing sometimes nondiagnostic (titers already elevated at "acute" sample). There is usually a mild interstitial pneumonia with alveolitis, but this quickly resolves.
- The secondary stage of disease is sporadic in occurrence and may be caused by repeated infection, hypersensitivity, or possibly some alteration of host cell surface antigen in the lung. In these cases, there is a profound interstitial pneumonia with bronchiolitis and bullous emphysema likely caused by the intense respiratory effort.

(4) Diagnostic plan and laboratory tests. The clinical signs of this infection are seldom sufficiently specific to establish a diagnosis. Demonstration of the virus by culture of lung tissue is seldom possible in field cases. However, if the "acute" sample is taken very early in the course of the disease, blood samples may demonstrate seroconversion, and direct immunofluorescence on nasopharyngeal swabs or fresh lung tissue from a recently infected animal may provide a definitive diagnosis.

(5) Therapeutic plan

- The primary stage of infection usually warrants no treatment because it is mild, and animals quickly recover unassisted. Because of the potential for secondary bacterial invasion of the lung, prophylactic antibiotics may be indicated (see Chapter 7).
- Those cattle with the secondary stage may benefit from administration of antihistamines, corticosteroids, or both. It is extremely important in such cases to minimize respiratory stress and limit any need for exertion. Despite this, the case fatality rate in the secondary cases is high.

(6) Prevention. There are several BRSV vaccines available commercially, alone or in combinations with other respiratory viruses (modified live). These appear to be effective against experimental challenge and also in reducing the incidence of respiratory disease of nonspecific etiology in the field.

f. Bovine malignant catarrh (BMC) is an acute, highly fatal systemic disease of cattle characterized by nasal, ocular (keratoconjunctivitis), and gastrointestinal lesions. This disorder is included in consideration of the upper respiratory tract because it also causes a rhinitis that must be differentiated from other respiratory tract diseases.

- Patient profile. Any age, sex, or breed of cattle can be affected. Although individual animals usually are infected, BMC can also occur in outbreaks. The disease shows the greatest incidence in late winter, spring, and summer.
- Clinical findings
 - There are several clinical manifestation of BMC, including the peracute, alimentary tract, and common "head and eye" forms. These forms appear to be gradations of the same disease.
 - In the head and eye form, there is a sudden onset of fever (41°C–41.5°C), severe dyspnea with obstruction of the nasal cavities caused by exudate from the superficial erosions on the mucosa and mucopurulent nasal discharge. Also evident are ocular discharge, eyelid swelling, blepharospasm, and scleral congestion. These animals exhibit extreme dejection. Characteristic and distinguishing signs also include peripheral lymphadenopathy and severe oral erosions.
- Etiology. The clinical signs can be caused by two different infectious agents: the acelaphine BMC virus (wildebeest-associated virus, AHV-1) or a sheep-associated BMC virus, probably similar to AHV-1.
- Therapeutic plan. The disease is almost invariably fatal and must be distinguished from the treatable and mild causes of upper respiratory infections of cattle, such as IBR, PI-3, and BRSV.

C. Pharyngitis, laryngitis, and tracheitis in sheep, goats, and swine. Although these conditions undoubtedly occur in sheep, goats, and swine, far less attention is given to these conditions in North American veterinary literature.

- Viral causes of pharyngitis and tracheitis are similar to those in cattle (e.g., respiratory syncytial virus has been reported). A more common cause in the small goat herd or sheep flock is traumatic pharyngitis, caused by drenching or balling gun injuries as a result of owner-administered medications.
- Pharyngitis in swine is reported in some outbreaks of pseudorabies (Aujeszky's disease) and is part of the manifestation of anthrax in this species.

III. LARYNGEAL OBSTRUCTION

A. Equine strangles. Strangles, also called horse distemper, occurs worldwide and historically caused major epidemics in cavalry horses. This remains an important disease of horses in developed countries because of the resulting disruption in management in brood mare or racing farms, time and expense of treatment, and unpleasant aesthetics of draining abscesses and purulent nasal discharges.

- Patient profile. The disease affects horses less than age 5 or 6 years, although all ages can be susceptible. Foals less than 3 months are most often unaffected, probably because of colostral protection.
- Clinical signs
 - After an incubation period of 1–3 weeks, affected horses suddenly develop depression, complete anorexia, fever (39.5°C–40.5°C), and serous nasal discharge.
 - The nasal discharge rapidly becomes copious and purulent, and horses show signs of severe pharyngitis and laryngitis, with reluctance to swallow and a soft, moist cough that appears painful. Possibly as a result of this pain, affected horses often stand with their head and neck extended and may appear dyspneic.
 - For 3 or 4 days, the lymph nodes of the throat region enlarge and are hot, painful, and initially firm in consistency. The lymph node enlargement may be sufficiently severe to cause obstruction to swallowing, dyspnea, and, in severe cases, death by asphyxia. Within 10 days, the swollen lymph nodes begin to weep serum and develop a soft spot from which they rupture (usually externally) and drain thick yellow material. Occasionally, these nodes rupture and drain internally into the pharynx. Often, the horse shows an improved attitude when the lymph nodes rupture and drain.
- Complications
 - Most animals recover within 3–6 weeks, but there can be a number of secondary complications. These include pneumonia secondary to aspiration with internal rupture or extension into the guttural pouches, causing guttural pouch empyema.
 - Very young foals can develop bacteremia or septicemia with joint infections and generalized lymphadenopathy.
 - The most common complication is metastatic strangles, or "bastard strangles," in which abscesses can spread to internal organ systems (i.e., lung, mesentery, spleen, brain) and cause subsequent localizing signs. Localized abscesses on the limbs can induce limb edema with the lower limb swelling three or four times its size. Finally, a delayed reaction due to immunologic sensitivity to the streptococcal protein can result in vasculitis, causing purpura hemorrhagica.
- Epidemiology
 - Outbreaks in susceptible animals often occur in cold wet weather, although movement and exposure to infected horses are also factors.
 - The causative organism is highly resistant in the environment, lasting up to a

month outside the host. Thus, contaminated water or feed buckets, or even objects such as blankets, brushes, and tack can be the source of infection.

c. The organism is known to persist in the pharynx of clinically normal, recovered horses for up to 8 months, and field experience suggests that such horses can be a source for new infections during this time.

4. Etiology and pathogenesis

- Strangles is caused by a β -hemolytic streptococcus, *Streptococcus equi*, which is present in the nasal discharges and draining abscesses of affected horses. This organism is not considered part of the normal nasal flora of the horse.
- The bacteria usually is transmitted by inhalation but can also be ingested. After incubating for 1–3 weeks, the organism causes acute pharyngitis and rhinitis. *S. equi* has M protein in its capsule, which is antiphagocytic and provides a means of avoiding normal defenses.
- From the mucosal surfaces, the organism moves by **lymph drainage** to local lymph nodes (submandibular and retropharyngeal) with subsequent abscessation at these sites.
- Strong immunity occurs immediately after infection and lasts 6 months to several years.

5. Diagnostic plan and laboratory tests

- In addition to clinical signs, cultures of nasal swabs or lymph node draining is **key to diagnosis**. Although other streptococcal species, such as *S. zooepidemicus*, also are readily found in nasal swab, and can give mild upper respiratory signs along with occasional lymph node abscessation, identification of *S. equi* is important because it requires rigorous control and quarantine measures.
- Routine hematology reveals neutrophilic leukocytosis, hyperfibrinogenemia, and anemia of chronic infection, but this is not specific to strangles.

6. Therapeutic plan

- Horses with early clinical signs (e.g., fever, anorexia, depression, pharyngitis, purulent nasal discharge)
 - Procaine penicillin G is the drug of choice administered at 22,000 IU/kg twice daily intramuscularly for 5 days or until all clinical signs are absent.
 - Tetracyclines** are also effective but should be avoided because of the risk of inducing colitis.
 - Trimethoprim-sulfadiazine** is an alternative to penicillin treatment, with its advantage being oral administration.
- Horses with lymph node **abscessation** require local treatment to enhance **mummification** and drainage of the abscesses.
 - Hot packs and poultices can be applied to the area of swellings several times daily. When the abscesses are mature, with the softening of a point of overlying skin, they can be lanced and flushed with 3%–5% povidone iodine.
 - Parenteral** antibiotics given after abscess formation tend to prolong rather than arrest disease. However, some veterinarians suggest treating all affected horses to reduce the risk of **more** animals becoming ill.
- Horses recently exposed to strangles with yet no clinical signs may benefit from antimicrobial therapy (e.g., **benzathine** penicillin) at the time of exposure and every 2 days thereafter until the end of the outbreak. This may prevent seeding of lymph nodes with the organism.
- Treatment** for secondary complications
 - For bastard strangles, long-term penicillin treatment (3–6 months) is required. Oral phenoxymethyl penicillin (110,000 IU/kg every 8 hours) is possible but very expensive. Thus, trimethoprim-sulfadiazine is usually the **drug of choice**.
 - Purpura hemorrhagica**, being an immunologic disease, requires glucocorticoids (dexamethasone) and supportive treatment such as leg wraps for the limb edema (see Chapter 9).
 - Guttural **pouch empyema** is treated locally by flushing the affected pouches with saline through the pharyngeal opening, often with the use of indwelling catheters for repeated treatment.

7. Prevention

- Management strategies
 - From the onset of clinical signs, it is critical to isolate affected animals because this highly contagious infection can spread through many of the young stock. Isolation for at least 6 weeks after start of signs is suggested, but based on field experiences, some veterinarians strongly suggest 8 months is a safer time frame. Clearly this latter recommendation must have the full cooperation and understanding by the client.
 - Because of the highly resistant nature of the organism, it is also imperative to thoroughly clean and **disinfect** stalls and grooming and feeding equipment, and burn the bedding from infected animals.
 - Vaccination against this organism provides only partial protection, with reduced severity and incidence of disease if and when it occurs.
 - Several commercial products are available and require a minimum of three doses at 2- to 4-week intervals, followed by annual boosters. Some products also induce muscle soreness and possible abscess formation, and for this reason vaccination should be administered in the pectoral muscles.
 - A vaccine containing a concentrated, purified M-protein **extract** of *S. equi* has been shown to reduce the rate of clinical disease by 50%.

B

Laryngeal obstruction in cattle

- Patient profile and history
 - Adult cattle
 - Adult cattle develop acute laryngeal obstruction most commonly as a result of laryngeal necrosis secondary to balling gun and drenching injuries. Laryngeal edema secondary to smoke inhalation also can result in signs of laryngeal obstruction.
 - Chronic laryngeal obstruction in adults is most often caused by **retropharyngeal** swelling, either from lymphadenitis or abscess, or a tumor in the throat latch region.
 - Calves, particularly those between 3 and 18 months, most commonly develop laryngeal obstruction as a result of the calf diphtheria (oral laryngeal **necrobacillosis**).
- Clinical signs
 - Laryngeal obstruction causes characteristic **inspiratory dyspnea** and **stertor**. There is also apparent excessive salivation caused by the reduced willingness for the animal to swallow as a result of the dysphagia induced by laryngeal inflammation.
 - There is anorexia, depression, and fever in calves with diphtheria and adult cattle with balling gun injuries that develop extensive cellulitis. In calves with diphtheria, there is invariably a characteristic foul necrotic odor to the breath.
- Etiology and pathogenesis
 - Inflammation of the larynx and pharynx depends on the inciting cause. For example, smoke inhalation may be a combination of chemical and thermal burn and, thus, be diffuse yet affect only the superficial **mucosa**.
 - Balling gun injuries often extend into the interstitial tissues around the pharynx and have extensive cellulitis from contaminating oropharyngeal inhabitants.
 - In calf diphtheria, the initial **mucosal** trauma from coarse feeds or shedding **teeth** allows invasion beyond mucosal tissues of *Fusobacterium necrophorum*, which is responsible for both the extremely foul odor to the calf's breath and severe toxemia.
- Diagnostic plan. The clinical signs and history often are sufficient to provide a working diagnosis.
 - Oral** examination with a speculum or an endoscope via the nares provides ready assessment of most of the structures of the pharynx and larynx.
 - For balling gun injuries, radiology of the pharyngeal region also can be valuable with the demonstration of **gas** or foreign matter in areas swollen with cellulitis.

5. Therapeutic plan
 - a. Most conditions benefit from parenteral antibiotics, such as sodium sulfadimidine 150 mg/kg for 2–3 days or procaine penicillin 20,000 IU/kg intramuscularly twice daily for 5 days.
 - b. In severely dyspneic animals, a tracheostomy should be performed as asphyxiation may occur before laryngeal swelling subsides.
 - c. Those animals that are unable to swallow require **parenteral** fluid therapy.
 - d. In cases of balling gun **injury** that are recognized at the time of occurrence and presumed to have caused extensive trauma, emergency slaughter should be considered as an economic alternative to treatment

C. Laryngeal obstruction in sheep, goats, and swine

1. Patient profile and **history**. Similar sporadic causes of laryngeal obstruction as found in cattle also can occur in small ruminants. In sheep, there also have been outbreaks of laryngeal obstruction reported resulting from necrotic laryngitis, with *Fusobacterium necrophorum* isolated from lesions.
2. Clinical signs include laryngeal stenosis, causing the characteristic **inspiratory dyspnea**. Also, regional lymphadenopathy and lung abscessation are seen.
3. Therapeutic plan. Treatment is similar to that described for cattle. Laryngeal obstruction in swine is most commonly seen as a result of encroachment of regional subcutaneous abscesses or abscessed lymph nodes.

STUDY QUESTIONS

DIRECTIONS: Each of the numbered items or incomplete statements in this section is followed by answers or by completions of the statement. Select the ONE numbered answer or completion that is **BEST** in each case.

1. Which one of the following statements regarding viral respiratory disease in horses is true?
 - (1) Although equine viral arteritis (EVA) is associated with conjunctivitis, limb edema, and respiratory signs, subclinical infections are most common, with aborting mares acting as the main carriers of the virus.
 - (2) Equine rhinopneumonitis virus infection caused by equine herpesvirus-4 (EHV-4) results in fever, conjunctivitis, and cough, whereas equine herpesvirus-1 (EHV-1) is associated with late-gestation abortion and neurologic disease in addition to fever, conjunctivitis, and cough.
 - (3) In addition to fever and the acute outbreak of cough, equine influenza virus can cause limb edema, myositis, and conjunctivitis in affected horses.
 - (4) Painful submandibular lymph nodes are a characteristic of rhinopneumonia.
 - (5) In horses, clinical signs of viral infection are seldom pathognomonic, and finding evidence of seroconversion is the only method of identifying many viruses.
2. A growing pig shows acute nasal discharge and rhinitis, followed by shortening of the snout and turbinate atrophy. Which one of the following statements is true?
 - (1) Inclusion body rhinitis is a major factor in the turbinate atrophy noted.
 - (2) The changes to the snout are typical of necrotic rhinitis (bulldog nose).
 - (3) *Bordetella bronchiseptica* infection followed by *Pasteurella multocida* infection is the most likely cause of these clinical signs.
 - (4) Infection by type A swine influenza virus damaged the nares and was followed by *Fusobacterium necrophorum* infection of the turbinates.
3. Equine herpesvirus-4 (EHV-4) is recovered from a group of horses on a brood mare farm following an outbreak of upper respiratory disease. Which one of the following statements is correct?
 - (1) Vaccination against equine **herpesvirus-1** (EHV-1) provides temporary but strong cross protection against EHV-4 infection.
 - (2) Most respiratory outbreaks in horses caused by herpesviruses are caused by EHV-4.
 - (3) Complications of EHV-4 infection include abortion and hind-end paresis.
 - (4) Differentiating EHV-4 infection from **EHV-1** infection can be done using currently available serologic tests for specific antibodies.
4. A 3-week-old Arabian foal that has been receiving treatment for pneumonia dies and adenovirus is recovered from the lungs as the only pathogen. Which one of the following statements is true?
 - (1) Serum obtained from this foal immediately prior to death can be used to confirm an immunodeficiency in this foal.
 - (2) The presence of presuckle immunoglobulin M (IgM) against adenovirus confirms that this infection was present at birth.
 - (3) Adenovirus is unlikely to cause significant disease in **non-Arabian** foals.
 - (4) The other foals on the farm of similar ages should be vaccinated against adenovirus.
 - (5) Because secondary bacterial involvement was not found, a fungal pathogen should be suspected instead of a primary viral agent.

5. Respiratory dyspnea and stertor are observed in an 8-month-old Hereford calf. The rectal temperature is 39.9°C, and the calf is depressed. Furthermore, a foul, necrotic breath odor is detected, and the calf appears reluctant to eat. What is the most likely diagnosis?

- (1) Calf diphtheria caused by *Fusobacterium necrophorum*
- (2) Balling gun injury sustained 24 hours ago
- (3) Enzootic pneumonia complicated by lung abscessation
- (4) A tooth root abscess with invasion by anaerobic bacteria
- (5) Papular stomatitis or bovine virus diarrhea (BVD)

6. Absence of passage of air in one nostril is a specific finding that may suggest which one of the following?

- (1) Ethmoid hematoma, in a horse with concomitant blood-tinged and malodorous nasal discharge
- (2) *Oestrus ovis* infection, in a sheep with concomitant bloody nasal discharge from the affected nares
- (3) Inclusion body rhinitis, in a pig with concomitant serous bilateral nasal discharge and sneezing
- (4) Ethmoid carcinoma, in a cow with concomitant bilateral bloody discharge and bulging of the facial bones
- (5) Granulomatous pedunculated masses caused by Rhinosporidiosis, in a horse with minimal to no concomitant nasal discharge

7. An owner reports the sudden onset of a harsh cough and bilateral ocular discharge in an 18-month-old steer. Other similarly aged animals in the herd are also similarly affected. Clinical examination reveals a rectal temperature of 40°C, a serous nasal discharge, swollen conjunctiva, and small necrotic plaques in the nares. What advice should be offered to the owner?

- (1) The affected animals need to be isolated from the breeding herd because they may be a source of an infection that can cause cerebellar hypoplasia or immunotolerance in calves.
- (2) To halt the spread of infection, an intranasal vaccine can be administered.
- (3) Signs are typical for *Pasteurella pneumonia*; affected calves need to be placed on antibiotics.
- (4) This is likely an upper respiratory virus but additional tests will be needed to identify it.
- (5) Treatment should include broad-spectrum antibiotics because lung infection is likely to occur.

8. A yearling Standardbred filly is receiving treatment in a clinic for clinical signs of strangles. *Streptococcus equi* is cultured from the nasal cavity. The owner wants to know if she will be able to successfully bring the filly back to the riding establishment she operates. Of the following comments, which one is correct?

- (1) A vaccine containing the antiphagocytic *S. equi* capsular antigen can be administered to the other horses to prevent them from developing the disease. Immunity is highly effective but short lived.
- (2) This filly can spread the organism to the other horses for at least 6 weeks after her clinical signs resolve.
- (3) Although isolation of this filly is important during the clinical stage, other horses at the riding school are likely to carry the organism as part of their normal flora, which can undergo rapid proliferation at times of high stress.
- (4) This filly will have strong immunity as a result of clinical disease and thus is unlikely to develop the disease hereafter.

ANSWERS AND EXPLANATIONS

1. **The answer is 2 [II A 3 b (3) (e)].** Equine viral rhinopneumonitis caused by equine herpesvirus-4 (EHV-4) and equine herpesvirus-1 (EHV-1) is associated with fever, conjunctivitis, and cough. In addition, EHV-1 infection may cause late-gestation abortion and neurologic disease. Stallions, rather than aborting mares, are the presumed carriers of the virus that causes equine viral arteritis (EVA). Although equine influenza may be associated with limb edema and myositis, conjunctivitis is not a recognized sign. Equine rhinopneumonia, a viral process, should not induce enlarged and painful lymph nodes; this sign is more consistent with *Streptococcus equi* infection. Some viruses can be cultured in the early stages; therefore, serology is not the only means of identifying some viruses.

2. **The answer is 3 [I D 1 b].** Atrophic rhinitis often occurs after infection of *Bordetella bronchiseptica*, which causes acute inflammation. *B. bronchiseptica* infection is then followed by *Pasteurella multocida* infection. Inclusion body rhinitis can cause acute nasal discharge but is not thought to be a component of atrophic rhinitis. Necrotic rhinitis is a cellulitis in the soft tissues around the snout and is thought to be caused by *Fusobacterium necrophorum* infection.

3. **The answer is 2 [II A 3 b (2) (b)].** More than 80% of respiratory outbreaks involving equine herpesvirus are associated with equine herpesvirus-4 (EHV-4). EHV-4 cannot be differentiated from EHV-1 using commonly available serologic methods. Equine herpesvirus-1 (EHV-1) has only 20% homology with EHV-4; therefore, vaccination against EHV-1 does not provide much protection against EHV-4. Abortion and hind-end paresis are complications associated with EHV-1 infection.

4. **The answer is 3 [II A 3 c].** Adenovirus only causes severe disease in foals with combined immunodeficiency, such as that observed in some Arabian foals that lack the ability to produce their own immunoglobulins and are severely lymphopenic. The foal has no ability to produce its own immunoglobulin M (IgM); therefore, sera from the foal at this age will likely contain immunoglobulins present from the dam's colostrum. The other foals

on the farm are unlikely to be at risk for such a severe, overwhelming infection because they probably are not immunodeficient. Adenovirus in this case can be the sole pathogen for pneumonia.

5. **The answer is 1 [II B 6 a (5) (d)].** The respiratory dyspnea, fever, and necrotic breath odor are characteristic signs of calf diphtheria. A balling gun injury sustained 24 hours previously would not be likely to produce an extremely necrotic breath odor so quickly. The breath odor of lung abscesses is consistent with that described here, but calves with enzootic pneumonia will not have dyspnea on inspiration. Dental problems would not be characterized by dyspnea. Papules and ulcers, not necrosis, are more likely to be associated with papular stomatitis or bovine viral diarrhea (BVD).

6. **The answer is 4 [I B 2 b].** Clinical findings of ethmoid carcinomas in cattle include bulging facial bones and the discharge of blood from the nostrils. The bloody discharge should not have a notable odor. The discharge is not usually blood-tinged; it is blood. Clinical signs are usually minimal.

7. **The answer is 2 [II B 6 a (7) (a) (i)].** The signs are typical of infectious bovine rhinotracheitis (IBR). An intranasal vaccine is available that can be used in the face of an outbreak because it stimulates the production of local interferon within 72 hours. The presence of the conjunctivitis and nasal plaques should allow the clinician to rule out bovine virus diarrhea and mucosal disease. An animal with *Pasteurella pneumonia* would not have the conjunctivitis and would be significantly depressed. In uncomplicated viral infections such as this one, antibiotics are not indicated.

8. **The answer is 2 [III A].** Shedding of the organism that causes equine strangles (horse distemper), *Streptococcus equi*, can persist for 6 weeks or longer after the clinical signs of infection resolve; therefore, the filly is at risk of spreading the disease to other horses at the horse establishment. A vaccine is available, but the immunity is not strong and offers only partial protection. *S. equi* is not part of the normal flora in horses. Immunity does occur with infection but is not permanent.